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THE "PIG-BACKED" OR ALCOHOLIC KIDNEY OF DRUNKARDS.

A CONTRIBUTION TO THE POST-MORTEM DIAGNOSIS OF ALCOHOLISM.[1]

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I WISH to invite attention to a certain peculiarity of the kidneys of drunkards. It concerns the gross anatomy and appearance of these organs, and chiefly their shape. A certain constant naked-eye appearance of the kidneys of hard drinkers on the autopsy table appears to me diagnostic of alcoholism. It is due to a chronic, venous, or passive congestion, leading to a cyanosis and sometimes to œdema of the kidneys, giving to these organs a peculiar swollen, rounded shape, which I am in the habit of designating as the "pig-backed" kidney. It differs essentially from the renal cyanosis of cardiac disease. It differs also from Bright's disease, and I believe it to represent an independent kidney affection.

While I have been pointing out this "pig-backed"

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kidney for some time in my laboratory demonstrations, and lately have repeatedly shown specimens of it in connection with other lesions of alcoholism before the Philadelphia Pathological Society, I have not as yet published a detailed account of this peculiar and fatal kidney affection. I am not aware that any of the authorities on renal diseases have studied or described this kidney lesion of drunkards, which I believe to be a quite interesting and significant one.

A word of comment upon the character of the material that has been under observation is necessary. I do not intend here to consider the general morbid anatomy of the kidneys of the universal "moderate" drinkers, since these individuals eventually die of any kind of ailments and with any kind of kidneys, often dying from exposure, carelessness, and irregularities in diet and life incident to the state of drunkenness, and perhaps not at all from the direct effects of alcohol.

The class of cases which I shall chiefly consider in this communication is one to which the coroner is more often called than the practising physician. I have selected only those cases where the person died more or less suddenly, where the history and general appearance clearly showed that death was due directly to alcoholism, and where all other factors in causing death had been carefully excluded as far as possible. I have particularly excluded such cases in which the persons had primary heart disease and other affections known to produce renal cyanosis. Most of the subjects had been in good general physical health, but having been inveterate drunkards and nearly always intoxicated, died while on a debauch, or, in rare instances, after a brief illness following it.

Through the kindness of the Coroner of Philadelphia who, of necessity, is called upon to issue death

certificates in such cases, I have had, during the last two years, the opportunity of making observations which some time ago numbered *two hundred and fifty cases*, belonging strictly to the category above referred to, and mainly from my own autopsies. In this city with a population of about one million, there are annually nearly two thousand cases of sudden death subject to legal inquiry. The proportion of cases of alcoholism appears thus remarkably large. I am indebted to my colleague, Coroner's Physician Stewart, for some of his autopsy material and his corroboration of the "pig-backed" kidney, and to Drs. George H. Chambers and A. Smith for valuable assistance in these observations.

A. Anatomical Considerations. I have met with two varieties of the kidney lesion now to be described:

First, a hard cyanotic form of the "pig-backed" or alcoholic kidney, in cases of sudden death; and

Second, a soft œdematous form of the same, in cases where death had been delayed.

First form: In nearly all persons who died more or less suddenly from the direct effects of alcohol, I found the kidneys to present the following appearances: The kidneys are always above the normal size; they are often from one-third to one-half larger, and are sometimes even double their usual size and weight. They are longer and thicker than normal, while their width is diminished, so that the natural, characteristic flat kidney, or bean-shaped form, is changed to a swollen, rounded, sausage-like or "pig backed" appearance or form. They are bluish-red or livid in color from being engorged to their utmost capacity with venous blood, reminding one of the rounded

enlarged appearance of animal kidneys when overfilled by some artificial injection mass. In most cases these "pig-backed" kidneys are elastic, but quite hard when freshly removed from the body; but unlike the cyanotic induration from heart disease, they gradually become softer, unless the renal vessels are at once tied to prevent escape of blood from them.

On section the cut surface presents the same dark red or livid appearance as seen in the cardiac form of renal cyanotic induration. While in the latter, however, the pyramids of the medullary substance are especially congested, in the alcoholic cyanosis the whole kidney substance is almost uniformly dark red. Dark blood oozes from the cut surface and blood extravasations can be often seen by the naked eye, principally below the capsules.

Second form: The other form of the alcoholic kidney referred to, was met with in persons who had died some time after a debauch (suffering from a few hours to a few days from delirium tremens previous to death). The kidneys of such subjects are found to be soft and flabby from œdema, and they are less red; otherwise presenting the same appearance as the hard variety of alcoholic renal cyanosis, including the "pig-back" shape of the kidneys.

In either form of this kidney lesion the capsule strips off easily, the surface is smooth, but often lobulated, not unlike the lobulation seen in congential conditions. If the alcoholic lesion is not complicated by heart disease, the "pig-backed" kidney is less heavy in proportion to size, and less firm and tough and also less dry than the kidneys of the pure cardiac renal cyanosis. This is probably due to the greater amount of blood present and to the accom-

panying œdema, which I have not seen in the cardiac variety.

In the purely cardiac affection, however enlarged, the kidneys retain their natural flat shape. This may be explained by the fact that in the latter case the morbid change is slow, gradual, and uninterrupted, and the resulting change (the induration from the connective tissue overgrowth) is a permanent one—whereas in the alcoholic cyanosis the process is rapid and intermittent, the effects of the alcohol being paroxysmal with each debauch and the mere resulting congestion receding more or less during the intervals of sobriety. It is probable that in the latter case the kidneys may return temporarily to their normal condition and shape.

A transverse section through the middle of the kidney from a case of alcoholic cyanosis is sometimes nearly circular, while a similar transverse section through a normal kidney or one of a cardiac renal cyanosis is oval or wedge-shaped and at least twice as long as broad.

Both kidneys are equally affected, sometimes the upper ends are found to be flattened or compressed anteriorly from pressure exerted by the enlarged liver on the right, and an enlarged spleen on the left side; the left kidney, as a rule, was more often found to be of a typical "pig-backed" shape than the right, and more frequently compressed at its upper end.

One hundred specimens of these kidneys were weighed and revealed the average weight of 250 grammes or a little over seven ounces—about two ounces over the normal average weight. The average variation in size and weight between the right and left kidney was insignificant.

A certain proportion of the 250 cases included in

my records showed, besides the alcoholic renal lesion, appearances of Bright's disease. It is difficult to draw in all instances a definite line between these two lesions, but I would say that in about 10 per cent. (or in 25) of the cases Bright's disease was evident. In 16 of these cases chronic catarrhal nephritis with formation of small cysts was noted; and in 9 cases chronic interstitial nephitis or a tendency to the granular contracted kidney was apparent. But, however affected otherwise, the "pig-back" shape of the kidneys was always a conspicuous feature in these cases. It appears that this chronic venous congestion (and œdema, when present) of alcoholism affects structurally diseased and normal kidneys in the same manner and manifests itself alike in both.

B. The cause of the "pig-backed" shape in the alcoholic renal cyanosis. The reason why the alcoholic kidney presents the roundish swollen shape is because the organs are over-distended with blood and serum, the kidneys acquiring the properties of, and in extreme cases may be compared with, true erectile or cavernous organs.

A "pig-backed" shape of kidneys can easily be induced experimentally in animals when the renal veins are tied or when kidneys removed from the body are artificially injected with a liquid.

Experiments have also shown that a hypertrophy of one kidney follows the removal of the other kidney, and under these circumstances I have seen the remaining kidney enlarge to double its natural size and acquire a typical "pig-backed" shape from being overfilled with blood.

A nearly similar appearance of hyperæmia of the kidneys may be seen in cases of acute fever, particu-

larly, in cases of infective fevers of children when death occurs during the height of the attack.

The loss of the flat kidney shape may be compared also with the loss of the biconcavity of red blood disks or corpuscles which when immersed in any liquid which they absorb become spherical bodies.

C. Microscopy. Sections were made from a number of specimens of the alcoholic kidneys, care being taken to examine microscopically all the different structural parts of these organs. For brevity's sake, only the essential changes will be referred to.

The cortical and medullary portions of the kidney appear to be similarly affected, but, as a rule, the latter less so than the former.

In the hard, cyanotic form of the alcoholic renal cyanosis the Malpighian glomeruli of the cortical portion are highly congested, and extravasated blood is seen within many of the Malpighian capsules; some are enlarged to double their normal size, but the majority appear rather compressed by the surrounding swollen uriniferous tubules and by the over-distended blood-vessels. The latter (both arteries and veins) show thickened walls and are deeply congested nearly everywhere throughout the organ. The stellate veins in particular are enormously distended and plugged up by blood corpuscles as if thrombosed. The lymph spaces beneath the capsules as well as around the tubules are also dilated and distended, and are either seen empty or they contain extravasated blood. In some sections the kidney structure gives the appearance of a cavernous change.

The epithelium of the convoluted tubules is cloudy, swollen, opaque, and the nuclei obscured by granules, which, however, become cleared up upon the addition of a solution of sodium hydrate. The epithelial cells appear to be double their normal size and

although no visible desquamation or proliferation of the cells could be noted in any one of the typical specimens examined, it appears that the lumena of the tubules are obliterated completely, as seen in transverse sections of the tubules.

In the straight tubules of the medullary portion the same changes have been noted, but are less marked.

The connective tissue elements of the pure alcoholic kidney show no pronounced hyperplasia. In the sections of most of the kidneys it is hardly perceptible. Sections in which the epithelial lining was forcibly removed by means of a camel's-hair brush showed the connective tissue to be dense, stiff, often pigmented, but only moderately increased in a few places around the bloodvessels.

The connective tissue increase was notably prominent only in sections derived from occasional specimens which showed at the same time other evidences of inflammatory changes (Bright's disease), and in cases complicated with heart disease. I do not remember having seen in one of the sections of the pure alcoholic affection tube-casts or blood within the uriniferous tubules.

In sections from the soft, œdematous form of the alcoholic kidney, the minute changes were essentially the same, but not so marked as those described above. Extravasations of blood were less frequently seen, while the connective tissue elements appeared occasionally more or less proliferated. In sections from some cases, however, no other change could be observed than the very prominent cloudy swelling of the epithelium peculiar to all the specimens of the alcoholic kidney.

I do not think it necessary to describe the sections

from the few cases in which Bright's disease and the alcoholic renal cyanosis coexisted.

D. Pathology. From the above studies it appears that the exact position of the "pig-backed" or alcoholic kidney in morbid anatomy is as follows: there are, as stated before, two forms or perhaps stages of this lesion. *First,* a *chronic or venous congestion with cloudy swelling,* or a renal cyanosis with hypertrophy represented by the *hard, red, "pig-backed" kidney* (the earlier and more active lesion); and *second,* a *hypertrophy with œdema* of the kidneys, represented by the *soft "pig-backed" kidney* (the more latent and less rapidly fatal lesion). The latter I met less frequently than the former, and believe it to be the later stage of the alcoholic kidney. Whether the lesion described eventually develops into Bright's disease, if the victim survives, I am unable to tell from my purely post-mortem observations.

I met with only twenty-five cases of established Bright's disease among two hundred and fifty cases of confirmed drunkards, from which it appears that Bright's disease is not more frequent in drunkards than in any other class of patients. Some of the foremost clinicians and authorities on kidney diseases (Bartels, Rosenstein, Tyson, etc.) are disinclined to give a prominent place to alcohol as a causative factor of Bright's disease, which appears to be in accord with the above observations.

That either form of the alcoholic renal cyanosis may be fatal in itself, without terminating in Bright's disease, I have satisfied myself, and hence I *believe that it deserves a place as a separate and independent kidney affection.*

The true mechanism of the *death* of drunkards from this alcoholic renal cyanosis is a matter of con-

jecture. If causes such as heart failure and œdema of the brain (which probably were the ultimate, immediate causes of death in some of my cases) are eliminated, a fatal uræmia is then the only plausible explanation. The empty urinary bladder in some cases, the high specific gravity of the urine, when found at all, post-mortem, in the bladder, and the histories which I had obtained in a few of the cases, favor such a view. In many cases of sudden death of drunkards it appears that death must have ensued under conditions similar to those of asphyxiation, because the blood in these cases was found dark and liquid throughout the system. A like condition of the blood, however, I have seen in certain other forms of poisoning, such as chloral, hydrocyanic acid, and carbolic acid. This subject requires further study.

But, while I have seen hundreds of drunkards dead of alcoholism, thousands of equally hard drinkers are at large and well. It appears that under favorable conditions the alcoholic renal cyanosis is capable of returning to the normal state of the kidneys, and that in its early stages it may be only a temporary lesion, paroxysmal, like the excessive drinking. Quite significant seems to me the experience of the drunkards themselves. One of them, a brewer, who had been a number of times prostrated from alcoholic excesses, and whom I treated about ten years ago for delirium tremens, told me that of late years he keeps himself well, because he stops drinking beer at once for a day as soon as he fails to urinate about every hour or two. I examined his urine lately and found no traces of renal disease.

E. Pathogenesis. The ordinary cyanotic induration of the kidneys, the result of long-continued permanent passive congestion of these organs, caused

principally and directly by organic heart disease and indirectly by aortic and pulmonary affections, has been recognized as a separate kidney affection since the time of Traube. Tumors pressing upon the vena cava or upon the renal veins, or thrombosis of the latter also produce renal cyanosis. So I should think any causes interfering with the return of blood from the kidneys will produce renal cyanosis, even if the cause resides in the kidneys themselves. The kidneys are organs especially predisposed to congestion, on account of their close and direct vascular communications with the large abdominal blood channels, and the renal veins, as Tyson well puts it, "are without valves and are the first, therefore, to receive the brunt of stagnation."

The alcoholic or "pig-backed" kidney we have seen to be essentially a renal cyanosis also, only differing, as pointed out above, in some anatomical features from the known cardiac renal cyanosis. The essential etiological factor, however, is in both the same, namely, the chronic venous congestion. The question is only as to the mechanism of the production of the alcoholic cyanosis. In alcoholism we must look to the kidney substance itself for the cause of the obstruction to the renal circulation, because there is, as a rule, no cause for it outside of the kidney: no disease of the thoracic viscera and no thrombosis of the veins. Of the latter fact I have satisfied myself from the examination, in a number of cases, of the vena cava and the renal veins. Besides, in most of my rapidly fatal cases of alcoholic cyanosis I found the blood to be non-coagulable as stated before. It is possible that the large, fatty liver, which sometimes reaches an enormous size, may be held accountable for the pressure upon the large abdominal venous trunks; but I have

often seen "pig-backed" kidneys in drunkards with but moderate hepatic enlargement.

I have heard Virchow say that "beer drinkers have *hypertrophied* kidneys." This view is also expressed by other pathologists, but no explanation is offered, nor have the anatomical peculiarities of the kidneys of drunkards been described. In relation to the pathogenesis of the alcoholic renal cyanosis the following explanation suggests itself:

It is well known that in persons who ingest great quantities of fluid, particularly alcoholic beverages in dilute form, the kidneys are strained to overwork. The quantity of urine normally passed by such persons is enormous so long as the kidneys act at all, and can at times be favorably compared with the quantity of urine passed in diabetes. It is evident that such overwork must invite a constant active (arterial) hyperæmia which of necessity produces a hypertrophy, due to a cloudy swelling of the epithelium and an overgrowth of all the structural constituents of the kidneys. A long continuance or a constant repetition of these conditions leads eventually to a passive (venous) congestion, which persists or increases or subsides according to the repetition and the duration of the debauches of the drunkard. The renal circulation of the blood is retarded by the pressure of the swollen epithelium of the uriniferous tubules as exerted upon the vessels —a pressure which the thicker walls of the intertubular arteries and arterioles can resist more readily than the soft and thin walls of the corresponding veins. There appears to be also thrombosis of the stellate veins. The exit of blood is thus retarded while the arterial pressure continues unabated and keeps the kidneys constantly overfilled with blood. Eventually the kidneys are filled to their utmost

capacity with blood overcharged with carbonic acid, and in consequence also with serum, which, leaking out of the obstructed veins into the renal lymph spaces, makes the kidney tissue œdematous; blood extravasates also, and adds to the firmness, redness, and roundness of the organ. Finally, the renal circulation comes to an entire standstill, coincident probably with a suppression of the urinary secretion.

This seems quite evident from the post-mortem appearances and from the swollen, cyanotic, and sometimes œdematous "pig-backed" kidney of drunkards, and is suggested also from the observation derived from the microscopicial examination of sections of these kidneys.

I will return to these studies in a future paper.

F. Statistical Remarks. Of the 250 cases of sudden death from alcoholism so far analyzed, 176 were men and 74 women. The hard, red form of the "pig-backed" kidney was found in men, principally in the younger and middle aged; while the softer œdematous form was more common in men of advanced age and in the women. The kidneys in which Bright's disease was coincident with the alcoholic lesion were mostly those of persons in advanced life.

The alcoholic renal lesion was nearly equally divided between persons of Anglo-Saxon and German descent, and, so far as I could perceive, equally between whiskey and beer and ale drinkers. I cannot speak about wine drinkers who in this country belong to the higher classes of society, and are seldom reached by the scapel of the pathologist.

It is interesting to note in connection with this point that the large, fatty liver (occasionally more or less indurated, but not contracted) which I met with indiscriminately in nine-tenths of all cases of death

from alcoholism was equally distributed between all the denominations stated above, including imbibers of strong and of dilute alcoholic beverages.

The following order in the occurrence of the important and more frequent lesions may be roughly given from my autopsy records of 250 persons who died from alcoholism strictly:

The "pig-backed" or alcoholic kidneys	248 times.
Fatty, infiltrated, enlarged liver	220 "
Cystitis, acute and chronic	170 "
Mammillated stomach (or chronic thickening of walls)	150 "
Congestion and œdema of brain (meningeal and ventricular)	150 "
Simple cardiac hypertrophy	90 "
Atheroma of vessels and valvular heart disease	50 "
Acute gastritis	50 "
Bright's disease (various forms)	25 "
Phthisis	20 "
Cerebral apoplexy	10 "
Cirrhosis of liver, with contraction below normal	6 "

I will make no comments upon the above figures in the present communication. The lesions of alcoholism other than the "*pig-backed*" kidney, I will consider on another occasion. Of great interest are the changes in the stomach, liver, and brain. Brief communications by myself upon some of the lesions of alcoholism may be found in the proceedings of the Philadelphia Pathological Society, 1885–86, when published.

The brains of confirmed drunkards should be studied by psychologists. The claim that chronic drunkenness is a form of insanity may perhaps be found to have some foundation.

G. The Medico-legal Aspect. A thorough knowledge and correct interpretation of the post-mortem appearances of alcoholism is perhaps to no end more important than when the life or welfare of a fellow-creature is at stake. There is a great deal to learn in this direction generally, and in no department of medical science are text-books more indefinite and "behind the times," and more rich in errors, than in the subject of medical jurisprudence.

I have myself once testified in court in an insurance case—and I know of other physicians who have done so—that "a certain person who had a large, fatty liver, to our best knowledge and belief was not likely to have been a hard drinker during life, chiefly because at the autopsy no cirrhosis of liver was found!" This was fully in accord with the teachings of our masters in medicine and with text-books. Yet, I am sorry to say, it is entirely wrong; and the jury decided against the insurance company on account of such erroneous medical testimony.

My experience has since taught me that cirrhosis with contraction of the liver is at least as rare an affection in drunkards as it is in "teetotalers," and that the traditional "hobnail" or "gin-drinker's" liver is not diagnostic at all, while the large, fatty liver is one of the most prominent signs of alcoholism. The facts are that in 250 drunkards I found the enlarged, fatty liver 220 times, and the contracted, cirrhotic liver but 6 times.

Often when a drunkard falls dead on any occasion, a cry of murder is raised, especially when bruises are found upon the body. It is here that a familiarity with the post-mortem appearances of *fatal* alcoholism is particularly necessary and nowhere

can ignorance on the part of the examiner do more harm than here.

The constant occurrence of the "*pig-backed*" or *alcoholic* kidney in hard drinkers who perished from their drinking, and the rarity of kidneys of such character in those who are not confirmed drunkards, induced me to regard the kidneys as a valuable sign in post-mortem diagnosis; and next to the presence of alcohol in the stomach, they are, together with the large, fatty liver, the most valuable proof that alcohol was operative or had contributed in producing death.

The "pig-backed" or alcoholic kidney, while a valuable diagnostic sign of the *effects* of the prolonged abuse of alcohol, may not show itself in persons who had been but moderately addicted to the use of alcohol, and in such case alcoholism should not be given as *the* cause of death.